lasting less than 30 minutes during the week before infarction. RV infarction was defined as ST-segment elevation of more than 0.1mV in lead V_{4R}. The PDCF was determined by simultaneous measurement of mean aorta pressure (Pa), coronary wedge pressure (Pw) assessed by pressure guidewire: PDCF=Pw/Pa. Sufficient collateral (group 1, n=10) was defined as PDCF index >24% and insufficient collateral (group 2, n=16) as PDCF≤24%.
Results: Baseline characteristics were similar between both groups. Preinfarction angina was detected less frequently in group 2 than in group 1 (29% vs 89%, p=0.004). RV infarction was observed more frequently in group 2 than in group 1 (71% vs 0%, p<0.001). Time to reperfusion was not related to PDCF.
Conclusion: Functional coronary collateral flow is responsible for the infrequency of RV infarction in patients with acute inferior MI.

1026-61 Quantitative Evaluation of Coronary Reperfusion by Transthoracic Doppler Echocardiography in Patients With Anterior Acute Myocardial Infarction

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Background: Although transthoracic Doppler echocardiography (TTDE) enables evaluation of distal left anterior descending (LAD) artery flow, its feasibility to evaluate coronary reperfusion in the acute phase before coronary intervention in patients with acute myocardial infarction (AMI) is not established.We hypothesized that presence or absence of TIMI 2 or 3 reflow can be evaluated by color TTDE of the distal LAD and good reflow with less Thrombolysis In Myocardial Infarction (TIMI) frame count by angiography is associated with preserved diastolic flow velocity of the LAD by pulsed TTDE. **Methods:** In 47 consecutive patients with a first anterior AMI within 12 hours from the onset, the presence of antegrade distal LAD flow and its diastolic peak velocity was evaluated by color and pulsed TTDE and compared with the TIMI frame count by subsequent coronary angiography. **Results:** 1) Coronary reperfusion was TIMI 2 to 3 in 23 of 47 patients. Diagnosis of TIMI 2 or 3 by the presence of distal LAD reflow by color TTDE had the sensitivity, specificity, and accuracy of 83%, 87%, and 85%, respectively. 2) In the 19 patients with visible LAD reflow by color TTDE, the diastolic peak velocity of the LAD flow by pulsed TTDE showed a significant and inverse correlation with the LAD TIMI frame count by angiography (r = -0.75, P = 0.0002). The diagnosis of TIMI frame count > 40, expressing poorer outcome, by the diastolic peak velocity ≤ 20 cm/sec with pulsed TTDE had a sensitivity, specificity, and accuracy of 80%, 93%, and 89%, respectively. **Conclusion:** TTDE enables noninvasive and quantitative evaluation of the coronary reperfusion in patients with anterior AMI in the acute phase prior to emergent coronary intervention.

1026-62 Distal Protection Device (PercuSurge) Can Reduce Infarct Size in Patients With Acute Myocardial Infarction Who Have Ruptured Culprit Plaque

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Background: Distal protection device is demonstrated to prevent distal embolism in the PCI of saphenous vein graft.However, its effectiveness in the PCI of native coronary artery is unknown. We examined its aspirated material and its effect on infarct size limitation in patients with acute myocardial infarction having or not having ruptured culprit plaque.
Methods: Consecutive patients (n=39) with acute myocardial infarction were treated by PCI using distal protection device (PercuSurge). Culprit lesion was observed by angioscopy and determined whether or not having ruptured plaque. Ruptured plaque was defined as a disrupted plaque with its content protruded and occupied >50% of lumen. TAT levels of aspirated blood were measured and compared between patients with and without ruptured plaque. Control patients were selected matching for gender, age, lesion site, occlusion time, and whether or not having ruptured plaque. Infarct size evaluated by peak-CK-MB, and resolution of ST segment elevation (ST-resolution) was compared between patients treated with distal protection device and their matched control.
Results: Ruptured plaque was detected in 56 patients. TAT levels were significantly higher in patients with ruptured plaque than in those without ruptured plaque (17.5±7.0 ng/ml vs. 8.3±3.0ng/ml, p=0.006). Infarct size evaluated by peak CK-MB (187±86 U/L vs. 357±232U/L p=0.007) and ST-resolution (66±15% vs. 40±21%, p=0.001) was significantly reduced by the use of distal protection device in patients with ruptured plaque, but not in patients without ruptured plaque (peak-CK-MB; 154±99 U/L vs. 175±116U/L p=ns: ST-resolution; 63±13% vs. 59±23%, p=ns). Aspirated material were detected more often (85.7% vs. 54.5%, p=0.038) in patients with ruptured plaque. Aspirated material was pathologically lipid-rich tissue or platelet and fibrin-rich thrombus.
Conclusions: Distal protection device reduced infarct size in patients with acute myocardial infarction who have ruptured culprit plaque. This result suggests that the infarct size is increased by the distal embolization of thrombus or lipid-rich material from culprit lesion.

POSTER SESSION

1043

Evaluating Risk Associated With Percutaneous Coronary Intervention

Sunday, March 07, 2004, 3:00 p.m.-5:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 3:00 p.m.-4:00 p.m.

1043-41 Baseline Heart Rate Predicts All-Cause Mortality After Percutaneous Coronary Intervention

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Background: Previous studies have demonstrated an association between resting heart rate (HR) and mortality. However, it is unknown whether revascularization reduces the risk associated with an elevated baseline HR. We sought to determine the impact of baseline HR on outcomes after percutaneous coronary intervention (PCI).
Methods and Results: We followed 4580 consecutive patients who underwent PCI at the Cleveland Clinic Foundation between 1997 through 2001. We excluded patients with cardiogenic shock, Class III or IV congestive heart failure (CHF), or recent myocardial infarction. The primary endpoint of the study was all-cause mortality. We divided our cohort into quartiles of baseline HR (see table). Baseline characteristics were similar between the quartiles except for a higher percentage of patients on beta blockers (BB) in quartile 1. After a median follow-up of 2.4 years, there were 400 deaths (8.7%). There was a significant increase in risk of death with increasing quartile of baseline HR (table). After controlling for confounders and BB use, baseline HR remained independently predictive of all-cause mortality (OR 1.18; 95% CI 1.1-1.2, p<.0001 for each 10-beat increase in HR). BB use was not an independent predictor of survival in this cohort.
Conclusion: Resting HR, a marker of autonomic tone, is an independent predictor of all-cause mortality after PCI. Further study is needed to identify therapies to attenuate the risk associated with autonomic dysfunction in patients with coronary disease.

	Q1 N=1146	Q2 N=1069	Q3 N=1265	Q4 N=1100	P
Mean HR	56 ±4	66 ±2	74 ±3	88 ±10	
% BB	43%	37%	35%	32%	<0.0001
Mortality	5.5%	7.1%	10.2%	12.0%	<0.0001

1043-42 A Comparison of the Effect of Eptifibatide Plus Heparin Versus Bivalirudin on Thrombin Generation and Clot Lysis in Patients Undergoing a Percutaneous Coronary Intervention

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Background: Both platelet rich thrombi (white clots) and fibrin rich thrombi (red clots) are involved in the pathophysiology of acute coronary syndromes and PCI complications. Eptifibatide, a GPIIb-IIIa antagonist, prevents the formation of platelet rich thrombi, whereas bivalirudin prevents the stabilization of platelet rich thrombi by fibrin. Bivalirudin is increasingly being used in the cath lab as a substitute for GPIIb-IIIa antagonists plus heparin. Previous studies reported increased thrombin generation following cessation of heparin, whereas no such increase occurred with GPIIb-IIIa antagonists. This study compared the effects of bivalirudin versus eptifibatide plus heparin on thrombin generation and clot lysis. **Methods:** Twenty patients scheduled to undergo PCI with either eptifibatide (10 patients) or bivalirudin (10 patients) were enrolled. Blood samples were collected at 5 intervals from baseline through 24 hours post treatment. ELISA analyses assessed levels of prothrombin fragment 1.2 (PF 1.2), fibrinopeptide A (FPA), and D-dimer. Platelet aggregation by light transmission aggregometry was measured through 4-6 hours. **Results:** PF 1.2 levels were unchanged in patients receiving bivalirudin, whereas those receiving eptifibatide and heparin demonstrated a slight increase in activity, likely due to cessation of heparin. FPA levels initially decreased with both treatments, then increased at 24 hours, indicating a resumption of thrombin turnover. D-dimer levels, a measure of clot lysis, were relatively unchanged with bivalirudin treatment, but increased over time with eptifibatide and heparin. ADP and TRAP induced platelet aggregation were effectively inhibited by eptifibatide, whereas no consistent inhibition was observed with bivalirudin. **Conclusion:** Neither bivalirudin nor eptifibatide increases thrombin generation, although the combination of heparin with eptifibatide increases thrombin rebound in some patients. Clot lysis does not appear to improve with bivalirudin to the extent observed with eptifibatide. This study suggests that eptifibatide combined with bivalirudin could maximize platelet inhibition, thrombin inhibition, and clot lysis.